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Cardiac Vagal Control and Depressive Symptoms: The Moderating Role of Sleep Quality

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Lower cardiac vagal control (CVC) has been linked to greater depression. However, this link has not been consistently demonstrated, suggesting the presence of key moderators. Sleep plausibly is one such factor. Therefore, we investigated whether sleep quality moderates the link between CVC (quantified by high-frequency heart rate variability, HF-HRV) and depressive symptoms (assessed using established questionnaires) in 29 healthy women. Results revealed a significant interaction between HF-HRV and sleep quality in predicting depressive symptoms: participants with lower HF-HRV reported elevated depressive symptoms only when sleep quality was also low. In contrast, HF-HRV was not associated with depressive symptoms when sleep quality was high, suggesting a protective function of high sleep quality in the context of lower CVC.

Low cardiac vagal control (CVC), typically assessed by heart rate variability related to respiration (respiratory sinus arrhythmia), has been linked with various mental health outcomes and particularly with diagnosis and symptoms of depression (e.g., Rottenberg, 2007). But while several empirical findings have supported an inverse relationship between CVC and depressive symptoms (e.g., Agelink et al., 2001), other studies have revealed no or even the opposite pattern of results (e.g., Lehofer et al., 1997). This suggests there might be moderators of the link

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between CVC and depressive symptoms. In the following, we will consider sleep quality as an important factor that may help explain the inconsistent links between CVC and depressive symptoms. Reduced sleep quality has emerged as an important risk factor in a variety of physical and mental health conditions including depression (see Imeri & Opp, 2009; Taylor, Lichstein, & Durrence, 2003), whereas high sleep quality may well have the potential to protect against elevated depressive symptoms in individuals with lower CVC.

CARDIAC VAGAL CONTROL AND DEPRESSIVE SYMPTOMS

CVC is linked to the functioning of one branch of the autonomic nervous system—the parasympathetic system—which is believed to subserve broad homeostatic functions (e.g., Berntson et al., 1997; Grossman & Taylor, 2007). The parasympathetic system influences heart rate via the vagus nerve, which originates in the brain stem and projects to many organs, including the heart. Respiratory sinus arrhythmia reflects the variation in heart rate that is related to the respiratory cycle and is thought to index CVC (e.g., Berntson et al., 1997).

Empirical evidence for the link between CVC and depression often supports a relationship between lower CVC and greater depressive symptoms (e.g., Licht et al., 2008; Rottenberg, 2007). Specifically, it has been shown that lower levels of resting CVC are related to higher depressive symptoms in participants with clinical depression (e.g., Agelink et al., 2001), remitted depression (e.g., Licht et al., 2008), and in healthy but high-risk population groups (Gentzler, Rottenberg, Kovacs, George, & Morey, 2011) as well as in healthy participants (Hopp et al., 2013). However, other studies have found no relationship (e.g., Gentzler, Santucci, Kovacs, & Fox, 2009; Lehofer et al., 1997) or even the opposite pattern showing, for example, higher CVC in depressed (vs. healthy) women (e.g., Thayer, Smith, Rossy, Sollers, & Friedman, 1998) or non-recovering depressed patients (Rottenberg, Wilhelm, Gross, & Gotlib, 2002). Overall, although the literature rather supports a negative link between reduced CVC and higher depressive symptoms, especially when including the meta-analysis by Rottenberg (2007), there are still studies showing positive links or no relationship between CVC and depressive symptoms. These findings suggest that there might be factors moderating the link between CVC and depressive symptoms.

SLEEP AS A MODERATOR OF THE LINK BETWEEN CVC AND DEPRESSIVE SYMPTOMS

One plausible moderator in the link between CVC and depressive symptoms is sleep. Individuals with severe sleep disturbances show a two- to fourfold risk for developing clinical depression (see Riemann, Berger, & Voderholzer, 2001). This points to the possibility for high sleep quality being a protective factor in the development of depressive symptoms. One longitudinal study provided support for this idea by showing that resilient young adults who did not develop depressive symptoms (despite low socioeconomic status) demonstrated shorter sleep latency and enhanced deep sleep (both indicators of better sleep) during childhood (Silk et al., 2007).

Although the exact functions of sleep remain unknown, there is increasing evidence that sleep facilitates self-regulatory as well as neurophysiological processes (e.g., Barber & Munz, 2010) in

order to support a more adaptive and flexible control of behavior, attention, and emotion (e.g., Walker, 2010). In contrast, poorer sleep quality may impair individuals' ability to regulate behavior and emotions by increased arousal (e.g., Riemann et al., 2010) and by disrupting higher cognitive functions which support self-regulatory processes such as cognitive emotion regulation (Mauss, Troy, & LeBourgeois, 2013). Consequently, for individuals whose reduced levels of CVC put them at risk for increased depressive symptoms by increased arousal and by impairing self-regulatory abilities, high sleep quality may provide additional regulatory resources and offset this risk. In other words, CVC and sleep quality might interact with one another, such that CVC is associated with higher depressive symptoms only in the presence of low but not high sleep quality.

Studies investigating the link between CVC and depressive symptoms have just started to take sleep quality into account, mainly in clinical samples (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014; Yang et al., 2011). These studies support the idea that sleep may play a role in the link between CVC and depressive symptoms by showing, for example, links between reduced sleep quality and reduced CVC in depressed but not in healthy individuals (Yang et al., 2011). One study directly tested the model we propose here, whereby CVC and sleep quality interact in predicting depressive symptoms. This study provided evidence for a moderating role of sleep in healthy children between the ages of 8 and 9 years (El-Sheikh, Erath, & Keller, 2007). It showed that low levels of CVC were only linked with greater depressive symptoms in the context of low sleep quality. To date, no study has investigated this relationship in adults.

THE PRESENT STUDY

The present study was designed to examine sleep quality as a moderating factor in the relationship between CVC and depressive symptoms. It did so by testing, in a generally healthy adult sample, the interaction between trait-level resting CVC and subjective sleep quality in predicting depressive symptoms. Because both CVC and sleep vary across the lifespan and between genders (e.g., Carrier, Land, Buysse, Kupfer, & Monk, 2001; De Meersman & Stein, 2007), this first study in adults involved only young women to reduce these sources of variance. We assessed CVC by quantifying heart rate variability in the high-frequency spectral band (HF-HRV; Berntson et al., 1997) during an extended resting baseline in order to enhance reliability. To standardize mental activity and ensure a neutral character of this baseline assessment, we used a minimally demanding task in the form of a neutral film (as recommended by Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). Furthermore, it has been recommended to use a second baseline measurement when assessing resting CVC to estimate its consistency across situations (Bertsch, Hagemann, Naumann, Schachinger, & Schulz, 2012). Therefore, we included a second baseline, the 2-min prefilm baseline (quiet sitting before the neutral film started) to confirm that this would provide the same results. We used established questionnaires to measure subjective sleep quality (Pittsburgh Sleep Quality Index; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and current depressive symptoms (German version of the Center for Epidemiologic Studies Depression Scale; Hautzinger & Bailer, 1993).

We expected subjective sleep quality to interact with CVC (as indexed by HF-HRV) in predicting depressive symptoms. Specifically, we expected lower CVC to predict higher

depressive symptoms only in the context of low sleep quality, but not in the context of high sleep quality, indicating a protective function of high sleep quality.

METHOD

Participants

Participants were 29 healthy female undergraduates (University of Salzburg, all Caucasian) aged between 19 and 31 years ($M = 23.6$ years, $SD = 3.3$) with a body mass index (BMI) between 17.4 and 31.7 ($M = 21.8$, $SD = 3.5$). The participants were non- or only occasional smokers with no known history of mental, neurological, or sleep disorders.

Procedure

The investigation took place as part of a larger study in the Clinical Stress and Emotion Lab of the University of Salzburg (for further details see Werner, Ford, et al., 2015; Werner, Schabus, Blechert, Kolodyazhnyi, & Wilhelm, 2015) including four visits to the lab (entrance examination and three nights in the lab). The whole study spanned 11 days. Participants were asked to complete daily sleep diaries to assure regular sleep cycles throughout the study. The study started with the entrance examination, during which participants completed several questionnaires, including the assessment of subjective sleep quality (referring to the prior four weeks), depressive symptoms (referring to the prior week), as well as general medical and psychological health condition. Specifically, physical fitness was rated on a 4-point Likert scale, 1 (*not very active*) to 4 (*physical activity three times a week or more*), and body mass index (BMI) was computed using participants' self-reported height and weight. The entrance examination was followed by three nights in the lab. The first night was used as an adaptation night. Night two and three included either the presentation of one completely neutral or one aversive film before participants went to bed; this was counterbalanced across participants.

The focus of the present investigation was the measurement of CVC (indexed by HF-HRV) during the presentation of the feature-length neutral film and the prefilm baseline, in relation to subjectively reported sleep quality and depression symptoms.¹ Participants were seated on a chair placed 40 inches in front of a 24-inch full HD monitor, and electrodes and sensors for measuring cardiovascular activity were attached. They were monitored by the experimenters, who were instructed to call the participants by name whenever they had the feeling that the participant would be drifting off. During film viewing (see description below), cardiovascular activity was recorded. Stimulus presentation of the film was controlled by E-Prime 2.0 (Psychology Software Tools, Inc., Pittsburgh, PA, USA). The study was approved by the local ethics committee. Participants signed written informed consent before the study and were compensated with course credit or payment of 100 Euro after the last session of the whole study period.

¹ Results concerning pre- to postsleep changes in emotional reactivity to the aversive film in relation to REM sleep, and regarding links between resting CVC (wake as well as sleep stage related) and sleep quality (subjective as well as objective variables) have been reported separately (Werner, Ford, et al., 2015; Werner, Schabus, et al., 2015).

Measures

HF-HRV

Psychophysiological measurements during the neutral film and the prefilm baseline were recorded using a 32-channel amplifier (TMSi, EJ Oldenzaal, The Netherlands) and the recording software package Polybench 1.22 (TMSi) with a sampling rate of 1024 Hz and 22-bit resolution. The recordings contained electrocardiogram (ECG), for which alcohol pads were used to clean the skin sites. ECG was recorded using disposable 55-mm diameter solid-gel snap electrodes; the electrodes were applied on the upper sternum and lowest rib on the left side. A 0.05 Hz high-pass filter was applied during ECG measurement. After recording, ECG raw data were bandpass filtered between 0.5 and 40 Hz and further processed using the software Autonomic Nervous System Laboratory (ANSLAB) version 2.51 (Wilhelm, Grossman, & Roth, 1999); R-spikes were determined automatically by ANSLAB and further manually checked. In ANSLAB, the preprocessed ECG was analyzed with power spectral analyses between .15 Hz and .40 Hz. More specifically, to obtain HF-HRV values, heart-period time series were linearly detrended and resampled at 4 Hz using cubic spline interpolation. Then, power-spectral densities for the whole duration of the neutral film (94 min) were computed using the Welch algorithm, which creates ensemble averages of successive periodograms. The averages were derived from spectra estimated for 120-s segments, overlapping by half. For each 120-s segment, we analyzed 512 points, which includes 480 sampled points with zero padding. We used Hanning-windowed segments that were subjected to fast Fourier transform. Estimates of power were adjusted to account for attenuation produced by the Hanning window, and distribution characteristics were normalized by natural-logarithm transformation. Although associations between vagal activity and HF-HRV might be influenced by variables like respiratory rate (e.g., Grossman & Taylor, 2007), we did not adjust for respiration, as this is less useful for individual difference analyses because people might differ in respiratory function due to factors unrelated to CVC, such as basal metabolic rate and respiratory pacemaker function (Grossman & Taylor, 2007). However, we did control for other possible confounding factors like age, BMI, and physical fitness. We used BMI as additional, interval-scaled proxy for physical fitness, as research often shows inverse links between BMI and physical fitness (e.g., Nikolaidis, 2013), which in turn can be linked to sleep quality and HF-HRV.

We used a neutral film to measure HF-HRV. We chose the neutral documentary *Living in a Monastery* (Spiegel TV, 94 min),² which describes the daily routine of nuns in a convent without any positive or negative emotional content. The film was validated in a pilot study with 11 female participants (age: $M = 21.8$, $SD = 2.0$) who provided valence ratings (1 = *positive*; 9 = *negative*) and arousal ratings (1 = *not arousing*, 9 = *very arousing*). Participants rated the film neutral to slightly positive ($M = 3.4$, $SD = 1.6$) and minimally arousing ($M = 2.5$, $SD = 1.5$). Additionally, we used the 2-min prefilm baseline (quiet sitting before the neutral film) as a comparison condition to enhance confidence in the results.

² This German documentary can be found on the Web page of the television channel *SpiegelTV* (<http://www.spiegel.tv/filme/kloster-leben-verliebt-gott/>).

Subjective sleep quality

We chose the Pittsburgh Sleep Quality Index (Buysse et al., 1989; German version by Riemann & Backhaus, 1996) to measure overall subjective sleep quality. The PSQI is a self-report questionnaire assessing sleep quality and sleep disturbances over the preceding 4-week time interval. Eighteen individual items generate seven “component” scores (each with values between 0 and 3): subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. These scores are summed up to obtain one global measure of subjective sleep quality (0–21). Lower values in the PSQI indicate better subjective sleep quality. The suggested clinical cutoff value for good subjective sleep quality in the PSQI is 5.

Depressive symptoms

To measure subclinical depressive symptoms in our healthy sample, we used the German version of the Center for Epidemiologic Studies Depression Scale (ADS: Hautzinger & Bailer, 1993; Hautzinger, Bailer, Hofmeister, & Keller, 2012; CESD: Radloff, 1977). This questionnaire has 20 items and refers to the preceding week. It includes emotional, motivational, somatic, and motoric-interactional complaints (e.g., “I did not feel like eating; my appetite was poor”; “I felt sad”) on a 4-point scale from 0 (*rarely or none of the time/less than 1 day during the last week*) to 3 (*most or all of the time/5–7 days during the last week*), which leads to a maximum depression score of 60. Higher scores indicate higher depression severity with values above 22 indicating clinically relevant depressive symptoms (Hautzinger et al., 2012). The German version of the CESD has demonstrated good internal consistency in the present sample ($\alpha = .89$) and has been shown to be highly correlated ($.72 < r_s < .94$) with other self-report questionnaires for depressive symptoms such as the *Beck Depression Inventory* (Hautzinger & Bailer, 1993).

Statistical Analysis

First, because most variables were non-normally distributed (*Kolmogorov-Smirnov*, *Lilliefors* $< .05$) or ordinally coded (subjective sleep quality, physical activity), we used Spearman correlations to investigate interrelationships between both HF-HRV variables and PSQI, depressive symptoms, as well as confounding variables (i.e., age, BMI, physical fitness). Only for the correlation of HF-HRV during the neutral film (HF-HRV film) with HF-HRV during the prefilm baseline (HF-HRV baseline) Pearson correlations were used. Second, we conducted linear regression models with HF-HRV, PSQI, and the interaction term between HF-HRV and PSQI (all independent variables were mean-centered) as separate predictors for depressive symptoms. This analysis remains appropriate given that it only requires normally distributed errors and homoscedasticity—which are present in the current data—and does not require normally distributed predictors (Field, 2009). Model 1 included HF-HRV film, whereas HF-HRV baseline was used in Model 2. We also report both models when including confounding variables as additional predictors.

Finally, we conducted an exploratory analysis to exclude the possibility that a mediation model accounts better for the data structure. We tested the indirect effect of HF-HRV on

depressive symptoms (i.e., CESD) via sleep quality (i.e., PSQI) using the PROCESS model (see Hayes, 2013). All analyses were run using IBM SPSS 22.

RESULTS

Preliminary Analysis

Descriptive statistics for HF-HRV variables, subjective sleep quality, and depressive symptoms are summarized in Table 1. The means of depressive symptoms and sleep quality were in the normal range. Three participants (10%) exceeded the clinical cutoff (> 22) of the depression scale, indicating clinically relevant depressive symptoms. On the subjective sleep quality scale, four participants (14%) revealed values above the suggested cutoff (> 5) for insomnia. Nevertheless, we included all participants in the analyses, as these values are only clinically relevant values, but no statistical outliers (> 3 SDs), so as to not artificially restrict variance in these variables of interest.

Table 1 also presents zero-order relationships among HF-HRV variables, sleep quality, depressive symptoms, and possible confounding factors (age, BMI, and physical fitness). The two HF-HRV variables were highly correlated, indicating transsituational consistency of HF-HRV. Lower HF-HRV for the film but not the prefilm baseline condition was significantly related to lower sleep quality (i.e., higher PSQI scores; however with low multicollinearity: $tolerance > .89$, $VIF < 1.12$). Lower HF-HRV for the film as well as for the prefilm baseline condition was significantly related to higher depressive symptoms. Lower sleep quality was also related to higher depressive symptoms. None of the possible confounding variables were significantly related to both HF-HRV variables, sleep quality, or depressive symptoms.

TABLE 1
Descriptives and Zero-Order Correlations for Study and Control Variables

	<i>M</i>	<i>SD</i>	<i>CVC film</i>	<i>CVC baseline</i>	<i>Sleep quality</i>	<i>Depressive symptoms</i>
Primary study variables						
CVC film (HF-HRV, ms ²)	8.17	1.02	—	—	—	—
CVC baseline (HF-HRV, ms ²)	8.44	1.14	.87***	—	—	—
Sleep quality (PSQI, 1–21)	4.10 (4)	1.37 (1–7)	–.39*	–.28	—	—
Depressive symptoms (CESD, 0–60)	11.31	7.76	–.55***	–.49**	.64***	—
Control variables						
Age (years)	23.55	3.30	.09	.01	–.07	.08
BMI (kg/m ²)	21.80	3.50	.13	.19	–.05	.01
Physical fitness (0–4)	2.66 (3)	0.90 (1–4)	.15	–.05	.05	

Note. $N = 29$. Pearson correlation for CVC film with CVC baseline; Spearman correlations for all other variables. For PSQI and physical fitness (ordinal-scaled) we also report the median and range in parentheses. Larger values in the PSQI indicate worse sleep quality. CVC = cardiac vagal control, HF-HRV = high-frequency heart rate variability, PSQI = Pittsburgh Sleep Quality Index, CESD = Center for Epidemiologic Studies Depression Scale (German version), BMI = Body Mass Index.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$.

Sleep Quality as Moderator in the Relationship Between HF-HRV and Depressive Symptoms

To test the hypothesis that subjective sleep quality acts as a moderator in the relationship between HF-HRV and depressive symptoms, we conducted linear regression analyses. Model 1 included depressive symptoms as dependent variable as well as HF-HRV film (HF-HRV during the prefilm baseline, in the following referred to as HF-HRV baseline, in Model 2), subjective sleep quality, and the interaction of the two as independent variables (all independent variables were mean-centered). HF-HRV and subjective sleep quality were entered in block 1, and the interaction was entered in block 2. The results of the regression analyses for both models are displayed in Table 2. In Model 1, lower HF-HRV film was marginally related to higher depressive symptoms, whereas in Model 2 this relationship was significant. In both models, lower sleep quality was significantly related to higher depressive symptoms. Importantly, in both models these main effects were qualified by a significant interaction; for the addition of the interaction term to Model 1 (block 2), the change in R^2 was significant, R^2 change = .11, $F(1, 25) = 6.06$, $p = .021$; this effect was even stronger for Model 2, R^2 change = .16, $F(1,25) = 10.15$, $p = .004$.

To illustrate the interaction, the relationship was plotted using mean values ± 1 standard deviation of subjective sleep quality and HF-HRV (see Figure 1; only the interaction of Model 1 was plotted as results in Model 2 were equivalent). Simple slope analyses revealed that for participants with higher sleep quality (low PSQI scores), HF-HRV did not predict depressive symptoms, $\beta = .06$, $t(28) = .31$, $p = .756$ (Model 2: $\beta = .01$, $t(28) = .08$, $p = .934$). However, for participants with lower sleep quality (high PSQI scores), lower HF-HRV significantly predicted higher depressive symptoms, $\beta = -.61$, $t(28) = -3.04$, $p = .005$ (Model 2: $\beta = -.78$, $t(28) = -3.96$, $p \leq .001$). Additionally, individuals with higher HF-HRV reported the same amount of depressive symptoms whether they reported high or low sleep quality, $\beta = .25$, $t(28) = 1.43$, $p = .165$ (Model 2: $\beta = .17$, $t(28) = 1.00$, $p = .328$), whereas individuals with lower HF-HRV reported significantly more depressive symptoms when they were low (vs. high) in sleep quality, $\beta = .92$, $t(28) = 4.14$, $p \leq .001$ (Model 2: $\beta = .96$, $t(28) = 4.94$, $p \leq .001$). These results

TABLE 2
Current Depressive Symptoms (CESD) as Predicted by Cardiac Vagal Control (HF-HRV Film and Baseline) and Subjective Sleep Quality (PSQI)

	<i>B</i>	<i>SE</i>	<i>CI (l; u)</i>	β	<i>t</i>	<i>p</i>
Model 1						
CVC (HF-HRV film)	-2.07	1.10	-4.33; 0.19	-.27	-1.88	.071
Sleep quality (PSQI)	3.33	0.84	1.60; 5.07	.59	3.96	.001
CVC x sleep quality	-1.85	0.75	-3.40; -0.30	-.35	-2.46	.021
Model 2						
CVC (HF-HRV baseline)	-2.61	0.91	-4.48; -0.73	-.38	-2.87	.008
Sleep quality (PSQI)	3.19	0.75	1.65; 4.74	.56	4.25	.001
CVC x sleep quality	-1.97	0.62	-3.24; -0.70	-.42	-3.19	.004

Note. $N = 29$. Adjusted R^2 for Model 1 = .48, for Model 2 = .56. CVC and sleep quality were always entered in step 1 of the regression analyses, whereas the interaction term was entered in step 2 to obtain separate statistical values for this addition to the model. HF-HRV and PSQI are mean-centered. For abbreviations, see Table 1.

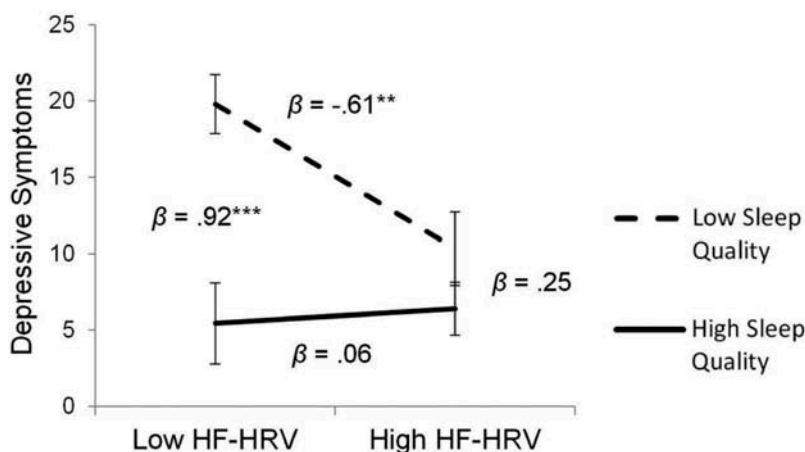


FIGURE 1 The interaction of high-frequency heart rate variability (HF-HRV during the neutral film) and subjective sleep quality (i.e., PSQI) on current depressive symptoms (total score on the German version of the Center for Epidemiologic Studies Depression Scale; CESD). Values show estimates at means ± 1 *SD* for HF-HRV film and sleep quality, and error bars represent corresponding standard errors. Asterisks denote slopes that are significantly different from zero ($*p \leq .05$, $**p \leq .01$, $***p \leq .001$).

indicate that participants reported higher depressive symptoms only when exhibiting both low HF-HRV and low subjective sleep quality.³

Next, we included age, BMI, and physical fitness as predictors in step one in the same regression analyses (Model 1 and 2). Although these variables revealed no significant correlations with our variables of interest, they are theoretically important and might have non-significant confounding effects on HF-HRV and sleep quality. When including age, BMI, and physical fitness in the analyses, the interaction of HF-HRV film and sleep quality became marginally significant (R^2 change = .08, $F[6, 28] = 4.00$, $p = .058$) whereas the interaction between HF-HRV baseline and sleep quality remained significant (R^2 change = .12, $F[6, 28] = 7.12$, $p = .014$).

Mediation Model

Given that HF-HRV film (but not HF-HRV during the prefilm baseline), sleep quality and depressive symptoms were associated with each other in zero-order correlations, a mediation of the HF-HRV-depression relationship by sleep quality would be a possible alternative way of interpreting the data. To test this possibility, we examined the indirect effect of HF-HRV film on

³ The pattern of results (in Model 1 and Model 2, for HF-HRV during the neutral film as well as the prefilm baseline condition) remained also unchanged when excluding the sleep-related item within the CESD. This was done in order to get a measure of depressive symptoms that was free of reduced sleep quality. Furthermore, one participant used allergy medications, known to have anticholinergic properties and thus potentially influencing HF-HRV assessment. We reanalyzed our data without this participant and the results remained the same. Lower HF-HRV during sleep and lower sleep quality (higher scores in the PSQI) were still significantly related to higher depressive symptoms. Most importantly, these main effects were qualified by a significant interaction; for the addition of the interaction term to the Model (in step 2), the change in R^2 was significant, R^2 change = .12, $F(1, 25) = 5.79$, $p = .024$.

depressive symptoms via subjective sleep quality. This indirect effect was computed using 95% bias corrected bootstrap confidence intervals, with 10,000 bootstrap samples. Results showed that the indirect effect was not statistically different from zero [95% CI: $-3.83 - .10$], Sobel's $z = -1.50$, $p = .132$, indicating that a mediation model did not provide a better fit to the data structure.

DISCUSSION

Although theoretical considerations suggest that lower CVC is linked to higher depressive symptoms, empirical evidence is mixed. This points to the existence of moderating factors. Therefore, the present study examined the effect of sleep quality as a moderator in the link between CVC (measured via HF-HRV) and depressive symptoms. Sleep quality is a plausible moderator because sleep quality (a) has a protective effect on depressive symptoms and (b) is thought to have those effects in part via a mechanism similar to that of CVC: self-regulation. We measured HF-HRV with two different assessments in order to enhance transsituational consistency of the effects of HF-HRV. Results for both HF-HRV assessments showed that sleep quality functioned as a moderator in the relationship between HF-HRV and depressive symptoms. Participants with lower HF-HRV showed elevated depressive symptoms only when they also reported lower sleep quality, whereas participants with lower HF-HRV did not show elevated depressive symptoms when they reported high sleep quality.

Because we found simple relationships among HF-HRV, sleep quality, and depression, we also tested a mediation model, wherein HF-HRV is linked to depressive symptoms via sleep quality. There was no statistical support for this model, which further bolstered confidence in the moderation model. These findings extend prior research in children (El-Sheikh et al., 2007) in emphasizing the important role of sleep quality as a protective factor in the relationship between HF-HRV and depressive symptoms.

Theoretical Implications: CVC, Sleep Quality, and Depressive Symptoms

Research on CVC and depression suggest that lower CVC is a physiological marker and risk factor related to the occurrence of increased depressive symptoms (e.g., Rottenberg, 2007). Recent theories explain these correlates of CVC within an evolutionary perspective linking CVC with arousal-related processes, such as adaptive behavioral strategies. In particular, it has been proposed that CVC is a concomitant of self-regulation and specifically emotion regulation in mammals (Porges, 2001; Thayer & Lane, 2000). Thus, they provide a biopsychosocial framework for interpreting CVC differences in, for example, mood disorders (Rottenberg, 2007). However, as empirical evidence is mixed, it has been suggested that moderating factors may influence the relationship between CVC and depressive symptoms (e.g., El-Sheikh et al., 2007; Yang et al., 2011).

We proposed that sleep quality may be a particularly plausible moderating factor, and the present results support this idea. Why might good sleep protect individuals from the risk associated with low CVC? One potential reason is that sleep provides additional resources for self-regulatory, and especially emotion-regulatory, processes, which are commonly impaired in depression (e.g., Hofmann, Sawyer, Fang, & Asnaani, 2012). Sleep disturbances and even mildly decreased sleep quality disrupt higher cognitive functions that may support emotion

regulation (Mauss et al., 2013). Moreover, sleep deprivation (an analogue to severe sleep disturbances) reduces functional connectivity between brain regions related to cognitive control and emotional responses (e.g., medial prefrontal cortex, amygdala; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). This may reflect decreased prefrontal control of emotions. Thus, it is possible that higher sleep quality may offset the risk for depressive symptoms imposed by reduced CVC, by facilitating emotion regulation.

Practical Implications

Our results provide support for a model regarding the development of depressive symptoms in which CVC and one's sleep quality are critically important. With a lifetime prevalence of around 16.2% (Kessler et al., 2003) and recurrence rates between 35% in the general population and 85% in specialized mental health facilities (Hardeveld, Spijker, De Graaf, Nolen, & Beekman, 2010), affective disorders (mainly major depression) belong to the second most frequent group of disorders (Wittchen & Jacobi, 2005). Therefore, early intervention in individuals with mild depressive symptoms—and prevention of major depressive disorder before it emerges—is of crucial importance for public health.

Although three participants exceeded the clinical cutoff of 22 points on the CESD, our study included mostly healthy participants with minimal to subclinical levels of depressive symptoms. Focusing on healthy participants (vs. clinical samples) provides valuable insights and extends knowledge on how to improve mental health before clinical disorders occur. It has been suggested that depression is best conceptualized on a continuum, and that clinical depression is not categorically distinct from milder forms of depressive symptoms occurring in generally healthy individuals (Rodriguez, Nuevo, Chatterji, & Ayuso-Mateos, 2012). Furthermore, it seems that even individuals with subclinical levels of depressive symptomatology can exhibit significant decrements in overall health, well-being, and daily functioning (Ayuso-Mateos, Nuevo, Verdes, Naidoo, & Chatterji, 2010). Thus, the present study has important implications for the prevention of clinically relevant depressive symptoms.

One set of practical implications of the present study concerns CVC. CVC can be improved via biofeedback (e.g., Nolan et al., 2005), leading to a reduction of depressive symptoms (Siepmann, Aykac, Unterdorfer, Petrowski, & Mueck-Weymann, 2008). CVC can also be enhanced by pharmacological and surgical means (Bernik et al., 2002; Singh, Kandala, & Camm, 2014). Interestingly, Tsai, Kuo, Lee, and Yang (2015) showed that slow-paced breathing (at a frequency of 0.1 Hz) can lead to increases in CVC as well, which improved sleep quality among individuals with insomnia. Furthermore, increasing physical exercise over a period of 6 months, which is very likely to increase CVC, has also been shown to have positive effects on sleep quality and depression as well as mood in general (Hartescu, Morgan, & Stevinson, 2015; Passos et al., 2011). The present study suggests that such efforts would be especially important in individuals with low resting CVC and low sleep quality.

A second set of practical implications concerns sleep. Our results suggest that improving sleep quality in individuals complaining about sleep problems is a crucial step for supporting mental health (Buysse, 2014). Higher sleep quality might buffer other risk factors like decreased CVC, at least in otherwise healthy individuals. Sleep interventions may be even more important than CVC interventions, as they are probably easier to implement. Research in clinical samples already showed that even short-term interventions (one session of cognitive behavioral therapy for insomnia) can improve sleep quality considerably and for at least 4 weeks after the

intervention (Ellis, Cushing, & Germain, 2015). Other studies using cognitive behavioral therapy for insomnia also demonstrated a reduction of depressive symptoms in patients with comorbid depression (e.g., Manber et al., 2008). Moreover, it has been suggested that sleep disruption plays an important role, functioning as a transdiagnostic characteristic of mental disorders and supporting the implementation of cognitive behavioral therapy modules for insomnia in the treatment of a variety of disorders (e.g., Harvey et al., 2014). The present results support the usefulness of such interventions, especially in participants with lower levels of resting CVC. However, as our results indicate that having either high sleep quality or high CVC can protect against greater depressive symptoms, treatment and preventive approaches may focus on improving one of these factors depending on feasibility of the specific treatment and individual preferences.

Limitations

This study has several limitations, including the use of a correlational design. Without investigating causal manipulations or longitudinal data it is not possible to fully ascertain any cause-effect assumptions in the examined models. Furthermore, the sample size is on the lower end for reliably testing moderation or mediation models involving three variables. In spite of this, the power for our whole moderation model (power = .99) as well as for the interaction term (power = .73) is similar to the power described in El-Sheikh et al. (2007), at least for HF-HRV during the prefilm baseline. It is also important to note that the present results replicated those reported by El-Sheikh et al. (2007), adding to confidence in them. Nevertheless, extending and replicating these findings using a longitudinal design and larger sample size (also including men and a broader age range to enhance generalizability) will be important. We focused on overall sleep quality and therefore used a well-validated self-report questionnaire for quantifying sleep quality, which correlates with several objective measures of sleep quality (Akerstedt, Hume, Minors, & Waterhouse, 1994). Nonetheless, it will be important to replicate the present pattern of results with objective measures of sleep quality. This will make a careful selection of objective sleep parameters necessary, as each single objective variable is not equivalent to overall subjective sleep quality (e.g., Blunden & Galland, 2014). Lastly, although we explicitly focused on a generally healthy sample to extend knowledge on how to improve mental health before clinical disorders occur, findings may not necessarily reflect relations between variables in adults with clinically significant depression or insomnia. Therefore, it is desirable to investigate this moderation model in clinical samples.

CONCLUSION

To our knowledge, this is the first study investigating the interaction between CVC and sleep quality in predicting mental health in a healthy adult population. Results demonstrated that CVC and sleep quality interact such that higher depressive symptoms were only reported when CVC (measured via HF-HRV) and sleep quality were both low. Reduced CVC seems to constitute a marker for elevated depressive symptoms only in the context of low sleep quality in healthy participants. Individuals' high sleep quality may offset the risk imposed by reduced CVC, consequently leading to decreased depressive symptomatology. These findings

support enhanced CVC as a resilience factor for mental health, but they also point to a protective function of high sleep quality for mental health in the context of reduced CVC.

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